	<b>Providing Anesthesia for Neurologically Deceased Donors (NDD)</b>	<b>Doc. No.:</b> ODHD-SRS.05.016
		<b>Rev.:</b> 03
		<b>Rev. Date:</b> 11-Dec-2020

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Providing anesthesia for neurologically deceased donors (NDDs) requires an understanding of the pathophysiology of cerebral herniation and brain death. Malignant intracranial hypertension and herniation is heralded by Cushing’s reflex with compromise of the pontine. With progressive loss of medullary function, patients often experience an “autonomic storm” with massive release of catecholamines and inflammatory mediators. The hemodynamic consequences of cerebral herniation are influenced by the rate of rise of intracranial pressure. The process of brain death may jeopardize end organ function and subsequent allograft procurement and performance. Priorities of care for NDDs are directed at resuscitating organs compromised by the evolution of brain death. Goals of anesthesia for organ procurement align with established ICU care of the NDD as well as abolishing spinal motor and hemodynamic reflexes.

**Equipment, Drugs and Anesthetic technique:**

- Neuromuscular blockade required to inhibit spinal motor reflexes
- Some advocate volatile anesthetics due to theoretical advantage of ischemic preconditioning
- Brain death may cause hemodynamic response to noxious stimuli; hypertension should be avoided

Below is a list of equipment and drugs that should be available for the procedure

**Equipment**

Intravenous infusion pumps	Bronchoscope	Gastric tube
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**Drugs**

Heparin 30,000 units	Long acting neuromuscular blocker
Vasopressin	Norepinephrine
Dobutamine	Milrinone
Dopamine	Labetalol
Nitroprusside	Esmolol
Mannitol	dDAVP
Furosemide	Insulin
Balanced crystalloid	Albumin
Blood products	Prostaglandin (provided by transplant team)

**Monitoring & Venous Access:**

- CAS monitors
- Urinary catheter
- Arterial & Central pressure monitors
- PA catheter or TEE for donors with reduced ventricular function (LVEF<40%)
- Two large bore peripheral IV catheters in upper extremities
- Hourly: arterial & venous blood gases, electrolytes, blood glucose, lactate & coagulation

**The ensuing section summarizes the clinical impact of brain death on major organ systems and includes recommendations for NDD management:**

**Cardiovascular**

- Autonomic storm = catecholamine cardiotoxicity with reversible left ventricular dysfunction
- Cerebral herniation = loss of sympathetic tone & systemic vasodilation, increased venous capacitance, relative hypovolemia exacerbated by prior mannitol therapy and central diabetes insipidus

**Management**

- Restore intravascular volume with balanced crystalloids or albumin
- Data suggests restrictive fluid resuscitation strategy (CVP 4-8) may increase yield of lung allografts without jeopardizing kidney function
- Synthetic colloids should be avoided due to potential osmotic nephrotoxicity

Vasopressin is the vasopressor of choice (0.01-0.04 Units/min)

- Limited evidence to justify preferential inotropic support: dobutamine, milrinone or dopamine are acceptable.

Standard hemodynamic goals include:

- MAP 70-90
- HR 60-120
- SBP 100-160
- SvO<sub>2</sub> ≥ 60%


**Pulmonary**

Common in NDD's

- Aspiration, neurogenic pulmonary edema and ALI/ARDS

**Ventilation**

- Avoid pulmonary derecruitment and ventilator induced lung injury
- Minimize disruption of breathing circuit
- Avoid high FiO<sub>2</sub>
- Use low tidal volumes (4-8mls/kg *Predicted Body Weight*) with modest levels of PEEP (10cmH<sub>2</sub>O)
- Periodic recruitment maneuvers
- Limit inspiratory pressures (<35cmH<sub>2</sub>O).

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### Renal

- Central diabetes insipidus common, contributes to hypovolemia
  - Treat with vasopressin (0.01-0.04 Units/min or dDAVP (1-4 mcg q6-8hrs iv/sc) when urine output >4mLs/kg/hr)
  - will assist in fluid management and avoid severe hypernatremia that can contribute to hepatic allograft dysfunction
- Urine output goal = maintain >0.5ml/kg/hr

### Hematologic


- Disseminated intravascular coagulation may complicate brain death
- Correct coagulopathy in actively bleeding donors
- Optimal hemoglobin concentration is unknown but likely between 70-100g/dL
- DIC is a contraindication to antifibrinolytics

### Endocrine

- Poikilothermia: results with brain death due to disruption of hypothalamic thermoregulation
- Pituitary function compromised (posterior>anterior) causing vasopressin deficiency with DI and reduced vasomotor tone
- Provide triple hormonal therapy (THT) to all donors (although cortisol levels may be normal)
 

*(Note: these treatments generally initiated in ICU):*

  - L-thyroxine (T<sub>4</sub>, Levothyroxine, Synthroid) 100 µg IV bolus followed by 50 µg IV q12h (administer only when serum K<sup>+</sup> > 3.5)
  - Vasopressin ≤ 2.4 units/hour (0.04 units/minute) IV infusion
- Biochemically, NDDs resemble a sick euthyroid state with reduced T3 levels
- Retrospective studies suggest that this THT increases the number of organs procured and improves allograft function
- Glycemic control: blood glucose levels <10

 <b>BC TRANSPLANT</b> <small>Provincial Health Services Authority</small>	<b>Providing Anesthesia for Neurologically Deceased Donors (NDD)</b>	<b>Doc. No.:</b> ODHD-SRS.05.016
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